treatment the facial paraesthesia had ceased. After a further two days sensation was normal in the right arm and the sensory level on the trunk had descended to T6 on the right and T8 on the left. Grip in the right hand had improved (grade 3-4) and he was again able to move his right leg at the hip and knee (grade 3) and ankle (grade 2). After 10 days' treatment he was able to walk with a single stick. Five weeks after the start of the treatment he was able to walk unaided, having only slight proximal weakness in the right leg. Power was normal in his right hand, though dexterity was impaired. Sensation was normal, with the exception of vibration sensation, which was impaired below the sternum. The plantar response was extensor on the right, and reflexes were brisk in the right leg.

Comment

Herpes-zoster myelitis is rare, and in their review of the published reports Rose *et al*[1] found only seven reported cases and added a case of their own. At necropsy their case showed extensive haemorrhagic necrosis of the spinal cord. The neurological symptoms and signs developed four days to six weeks after the herpetic rash, and the myelitis progressed over hours, days, or weeks. Four of the patients had died while the others recovered with variable neurological deficits. Although an autoimmune response had been proposed as the process causing central nervous system damage after herpes-zoster infections,[1 2] Hogan and Krigman[3] subsequently showed direct invasion of the spinal cord by herpes-varicella virus in a case of ascending myelitis.

In our patient symptoms and signs of myelitis progressed inexorably for four weeks until treatment with vidarabine was begun. Vidarabine is an antiviral agent which has proved valuable in the treatment of herpes-simplex encephalitis and in herpes-zoster infections in immunocompromised patients.[4] Its use has not been reported in herpes-zoster myelitis. Within days of starting the treatment our patient began to improve, and within five weeks he could walk unaided. Although herpes-zoster myelitis may improve spontaneously,[1] we think that the improvement was so dramatic after the start of treatment that it was not a mere coincidence. We therefore suggest that a parenteral antiviral agent should be given as soon as zoster myelitis is diagnosed.

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Gastric mucus secretion in ranitidine-treated patients

Ranitidine is a new H_2-receptor antagonist which at a dosage of 200 mg daily is reportedly as effective as cimetidine in the short-term treatment of duodenal ulcer.[1] Despite the remarkable antiulcer properties of cimetidine, this drug alters the composition of gastric mucus,[2-3] which apparently influences the rate of ulcer recurrence when treatment is stopped. The quality of gastric-soluble mucus appears to affect duodenal as well as gastric mucosal defences. Maintenance treatment with carbenoxolone after healing of duodenal ulcers by cimetidine is associated both with return to normal of the gastric mucus and with a comparatively low relapse rate once treatment is stopped. In contrast patients healed and maintained with cimetidine whose gastric mucus is of persistently poor quality show a significantly higher rate of ulcer recurrences.[3]

Acute intravenous infusion of ranitidine failed to affect pentagastrin-stimulated output of N-acetyl-neuraminic acid, suggesting a lack of effect on mucus secretion.[4] Nevertheless, the influence of prolonged ranitidine administration on gastric mucus has not been investigated. Indeed, the effect of a single dose of the drug cannot be taken to predict the behaviour of mucus in ranitidine-treated patients. While gastric mucus secretion is unaffected by a single intravenous dose of cimetidine,[4] appreciable alterations in the mucus properties are detectable after prolonged oral administration.[2-3] We have studied the behaviour of gastric mucus in patients given ranitidine for one month.

Patients, methods, and results

Twelve outpatients aged 26-73 years with endoscopically proved duodenal ulcer were given ranitidine 150 mg twice daily for four weeks. Treatment was then stopped and all patients subjected to endoscopy. Neutral and acid mucoproteins in fasting gastric juice were measured before and after treatment and a "mucoprotective index" calculated as: $(B/(A+B)) \times 100$, where A = acid mucoprotein complex (g/l) and B = neutral glycoprotein complex (g/l), the latter containing the bulk (75·2%) of carbohydrates.[2 3]

After the four weeks of ranitidine endoscopy showed complete healing of the ulcers in 11 of the 12 patients. No significant changes in mucoprotective indices were found after ranitidine (mean values before and after treatment 69·9 ± SEM 1·23 and 69·8 ± 0·9 respectively; $p > 0.05$ (Student's t test for paired data)).

Comment

Our results show that, unlike cimetidine, ranitidine does not influence gastric mucus secretion. This suggests that the cimetidine-induced alterations in gastric mucus are not related to the H_2-receptor blockage but are due to the drug itself. Differences between the two drugs have been reported. Aminopyrine metabolism, for example, is altered by cimetidine but is unaffected by ranitidine.[5] Indeed, ranitidine appears to be structurally different from cimetidine, being a substituted furan derivative with no imidazole ring; and this may account for different behaviour of the two drugs in many respects. Whether the lack of damaging effects on gastric mucus by ranitidine will result in a lower rate of ulcer recurrence in healed patients remains to be determined.

Ranitidine was kindly supplied by Glaxo SpA, Italy.

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Primary systemic amyloidosis presenting as extreme hyperlipidaemia with tendon xanthomas

Hyperlipidaemia is not a recognised complication of amyloidosis in the absence of nephrotic syndrome. We describe a patient with primary systemic amyloidosis in whom tendon xanthomas associated with extreme rise in plasma remnant and low-density lipoproteins were presenting features and preceded detectable renal disease.

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Case report

A 43-year-old man presented in 1977 with abdominal discomfort and chest pain on exertion. Physical examination showed hepatomegaly, thickening of both Achilles tendons, and xanthomas of the extensor tendons of the hands. Plasma cholesterol concentration was 11·5 mmol/l (444 mg/100 ml), and plasma triglyceride concentration was 2·4 mmol/l (212 mg/100 ml). Plasma lipid concentrations in parents and siblings were normal. Alcohol intake was minimal. Serum alkaline phosphatase and γ-glutamyl transpeptidase activities were raised (35 KA U/dl and 226 U/l). Plasma albumin, bilirubin, urea, and thyroxine concentrations were normal. Blood glucose concentration was normal. There was no rise in serum IgA, IgG, or IgM concentrations; no monoclonal immunoglobulin was seen on electrophoresis.

TABLE I—*Plasma lipoprotein lipids, plasma albumin concentration, and urinary protein excretion in patient with primary systemic amyloidosis*

Date	Plasma cholesterol (mmol/l)	Plasma triglyceride (mmol/l)	Lipoprotein lipids (mmol/l)		Plasma albumin (g/l)	Proteinuria (g/d)
			Cholesterol	Triglyceride		
1 August 1977	11·5	2·4			51	None
14 October 1977	12·2	3·4	V 2·5	1·6	47	None
			L 8·9	1·5		
			H 0·8	0·3		
8 December 1977	13·7	4·2			47	None
2 March 1978	12·3	7·2			40	2·3
24 April 1978	13·8	3·8			39	3·4
27 June 1978	14·7	3·9			38	
26 October 1978	19·0	8·1	V 7·6	5·8	36	
			L 10·9	1·9		
			H 0·5	0·4		
15 December 1978	19·0	8·0			34	6·2
20 June 1979	18·0	8·4	V 5·3	5·0	31	
			L 12·5	3·1		
			H 0·2	0·3		
31 July 1979	27·9	5·7				

V = Very low-density lipoprotein; L = Low-density lipoprotein; H = High-density lipoprotein.
Upper 95th centiles for men aged 40-69 years (see ref 2): VLDL cholesterol, 1·18 mmol/l; VLDL triglyceride, 1·56 mmol/l; LDL cholesterol, 5·06 mmol/l. Lower 95th centile of HDL cholesterol, 0·54 mmol/l.
Conversion: SI to traditional units—Cholesterol: 1 mmol/l ≈ 39 mg/100 ml. Triglyceride: 1 mmol/l ≈ 9 mg/100 ml.

Full blood count was normal. The bone marrow showed a small increase in apparently normal plasma cells, with normal erythropoiesis and granulopoiesis. No lytic lesions were seen in skeletal radiographs. At this stage there was no proteinuria. Findings on coronary angiography showed narrowing of the left main artery; other coronary vessels were normal. Amyloid deposits were seen in hepatic and myocardial biopsy samples on which basis primary systemic amyloidosis was diagnosed.

Plasma lipoprotein fractionation showed raised concentrations of low-density lipoproteins (LDL: density 1·006-1·063 g/ml) and of cholesterol-enriched very low-density lipoproteins (VLDL: density < 1·006 g/ml); plasma high-density lipoprotein (HDL) cholesterol concentration was low (see table). The presence of the lipoprotein of cholestasis (lipoprotein X) was excluded by agar-gel electrophoresis. LDL apoprotein B turnover, measured using ^{125}I-labelled autologous LDL,[1] gave a production rate of 16 mg/kg/day and a fractional catabolic rate of 0·177 per day (normal values, mean ± SD, 7·7 ± 1·9 mg/kg/day and 0·31 ± 0·04 per day[1]).

Progression of the disease over two years was associated with the development of restrictive cardiomyopathy, albuminuria, and hypoalbuminaemia, despite treatment with colchicine and chlorambucil. During the later stages λ light chains became detectable in the urine by immunoelectrophoresis. Plasma cholesterol concentration increased continuously, reaching 27·9 mmol/l (1077 mg/100 ml); plasma triglyceride concentration reached 8·4 mmol/l (743 mg/100 ml); and HDL cholesterol concentration decreased to 0·2 mmol/l (see table). Plasma lipoproteins showed no response to a fat-modified diet, clofibrate (2 g/day), or cholestyramine (12 g/day). The patient died in 1979. There was no autopsy.

Comment

The presence of high concentrations of cholesterol-rich VLDL in plasma reflects the intravascular accumulation of the triglyceride-depleted remnants of chylomicrons and VLDL.[2] The co-existence of this abnormality with a raised LDL concentration is a rare form of hyperlipidaemia. Recognised causes are myxoedema and the nephrotic syndrome.[2] In addition, we have seen a similar lipoprotein pattern in a patient with coincident familial hypercholesterolaemia and familial apolipoprotein E3 deficiency.[3] Each of these possible causes was excluded in the patient in this study. Although the rise in plasma lipids that occurred during 1978-9 may have been due to the development of nephrotic syndrome, both the hyperlipidaemia and the xanthomas preceded the onset of albuminuria by one year.

Our isotopic studies indicated that the increase in LDL concentration was due partly to overproduction and partly to impaired catabolism of the lipoprotein. No measurements were made of VLDL metabolism, but the abnormal lipid composition suggested a defect in the clearance of VLDL remnants.[4] The mechanism of these changes in lipoprotein metabolism is not clear. One possibility is that they were secondary to amyloidosis of the liver, which appears to have a role in the catabolism of both VLDL remnants and LDL.[2] It is also possible that lipoprotein catabolism was impaired by the formation of immune complexes, as can occur in multiple myeloma.[5] Whatever the underlying mechanism(s), it appears that primary systemic amyloidosis should be considered in xanthomatous hyperlipidaemia of obscure cause.

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